Intrathecal phenol

Intrathecal phenol (IP) was first reported to reduce pain and spasticity in the 1950s

However, complications such as bladder and bowel incontinence, limb weakness, and painful paraesthesia were highlighted, although it was not clear if these complications resolved or persisted.

Furthermore, the effect of IP seemed to diminish over time with pain and spasms returning

These issues, together with the introduction of intrathecal baclofen (ITB) in the late eighties and more recently botulinum toxin, have contributed to the relative neglect of IP as a management option. However, in appropriately selected people it may be a highly effective and well tolerated treatment in the management of severe spasticity and associated pain. The primary effect of phenol on nervous tissue is coagulation and denaturing of proteins, which leads to cellular and axonal damage.

Initially it was postulated that IP might act selectively on spasticity and pain without affecting the nerves responsible for bladder and bowel function.

However, it became apparent that phenol damages axons non-selectively, irrespective of their size, thereby affecting motor and sensory nerve fibres indiscriminately.

The magnitude of the effect depends on the volume and concentration of phenol used. Solutions of between 5%–8% phenol produce a relatively neuroselective effect whereas higher concentrations can cause extensive fibrosis and thickening of the arachnoid mater within the subarachnoid space.10 Administering phenol in glycerin renders it hyperbaric and viscous, resulting in limited spread, and hence localised nerve injury [1)].

1)
Jarrett L, Nandi P, Thompson AJ. Managing severe lower limb spasticity in multiple sclerosis: does intrathecal phenol have a role? J Neurol Neurosurg Psychiatry. 2002 Dec;73(6):705-9. PubMed PMID: 12438474; PubMed Central PMCID: PMC1757337.

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